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Editorial

Introduction to the review articles by ICADA on the pathogenesis of atopic dermatitis in dogs

In 2001 the then American College of Veterinary Dermatology Task Force on Canine Atopic Dermatitis published a comprehensive review covering what was then known about the pathogenesis of the atopic disease in dogs.¹ In the years following this landmark publication the committee evolved into 'The International Committee on Allergic Diseases of Animals' (ICADA; www.ICADA.info), which has been actively publishing further reviews of allergic disease in animals.² The purpose of the following collection of review papers is to provide an update to the original 'Task Force' review articles on canine allergic dermatitis, as much has been learned in the last 13 years. This update was prepared by searching online databases and abstracts from international veterinary dermatology meetings and congresses from 2001 to 2013 for articles relating to allergic and/or atopic diseases in dogs. Older works were also included when appropriate to provide background information and historical perspective. The manuscripts developed from this search were subsequently submitted to the entire ICADA membership for review and commentary prior to submission to this journal for a final peer review.

The six manuscripts in this collection are: clinical and histological manifestations of canine (AD); lymphocytes, cytokines, chemokines and the Th1/Th2 balance in canine AD; innate immunity, lipid metabolism and nutrition in canine AD; the role of antibodies, autoantigens and food allergens in canine AD; pathogenesis of canine atopic dermatitis: skin barrier and host-microorganism interaction; and the role of genetics and the environment in the pathogenesis of canine AD.

Since the 2001 publication, there has been a revision of the terminology used to refer to allergic diseases in dogs.³ Canine AD is currently defined as 'a genetically predisposed inflammatory and pruritic allergic skin disease with characteristic clinical features associated with IgE antibodies most commonly directed against environmental allergens'.³ Canine atopic-like dermatitis (ALD) is an 'inflammatory and pruritic skin disease with clinical features identical to those seen in canine atopic dermatitis in which an IgE response to environmental or other allergens cannot be documented'.³ This condition may be similar to 'intrinsic atopic dermatitis' (IAD) in humans, which refers to patients with symptoms clinically indistinguishable from AD but in which elevated allergen-specific IgE or demonstrable immediate hypersensitivity to allergens cannot be documented.⁴ Unfortunately, in dogs it is not known whether this perceived lack of detectable allergen-specific IgE reactivity is truly associated with the absence of IgE-mediated disease or simply reflects a failure to test for relevant allergens. As will be discussed in more detail, dogs do not show a consistent elevation in

allergen-specific IgE levels with atopic dermatitis and total IgE levels can be highly variable even in non-atopic dogs.⁵ In addition, healthy, clinically non-atopic dogs can have skin and serum reactivity to environmental allergens.^{6–10} Because canine AD and ALD cannot be definitively differentiated clinically (and in many of the studies reviewed here the two are either not separated or not specified) the two diseases will be collectively referred to as canine AD throughout these papers.

These review papers summarize the increasing amount of new knowledge in the subject of atopic dermatitis in dogs. However, there is still very much to learn. While the overall knowledge of the histopathology associated with canine AD has not changed much over the past few years, new studies have further defined the clinical phenotypes associated with AD. Great strides have also been made in further understanding the immunopathogenesis of AD in humans and in dogs. The cells of the innate immune system play much larger roles than we had previously suspected, while the role of total or allergen-specific antibodies appears to be smaller than previously thought. Although the central player in immunopathogenesis remains the T cell, new work has shown that the old Th1/Th2 paradigm is greatly oversimplified. We now also understand much more about the important role the epidermal barrier plays in the skin, both in health and in disease. Even so, there is controversy about whether the barrier dysfunction that has been described in canine AD is its cause or its effect. Studies to determine the role of genetics in the pathogenesis of canine AD have provided us with some information, but may have triggered more questions than they have answered, largely due to the complex interactions of genes with the environment and immune system of individuals. The development of large health records databases (such as those kept by pet medical insurance companies) can facilitate the identification of environmental risk factors, but mining of these databases is still a fairly new approach in veterinary medicine. Finally, we have learned much about the interactions and overlap between canine AD and other conditions that we previously considered to be entirely separate (such as adverse food reactions), but these interactions still remain imperfectly understood.

In conclusion, these articles represent a concise review and summary of the most important papers on the pathogenesis of canine atopic dermatitis in the last 13 years. It is hoped that this collection of articles will lead to better prevention, diagnosis and treatments of this disease for our canine friends as well as help to increase knowledge of other hypersensitivity disorders common in many species of animals, including humans. Much has been learned, but there is still so much to discover, and it is our hope that this collection contains the springboards for future research in the amazing and exciting topic of allergic skin disease.

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